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6/6/12

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OBSERVED IN TYPHOID FEVER.**

BY

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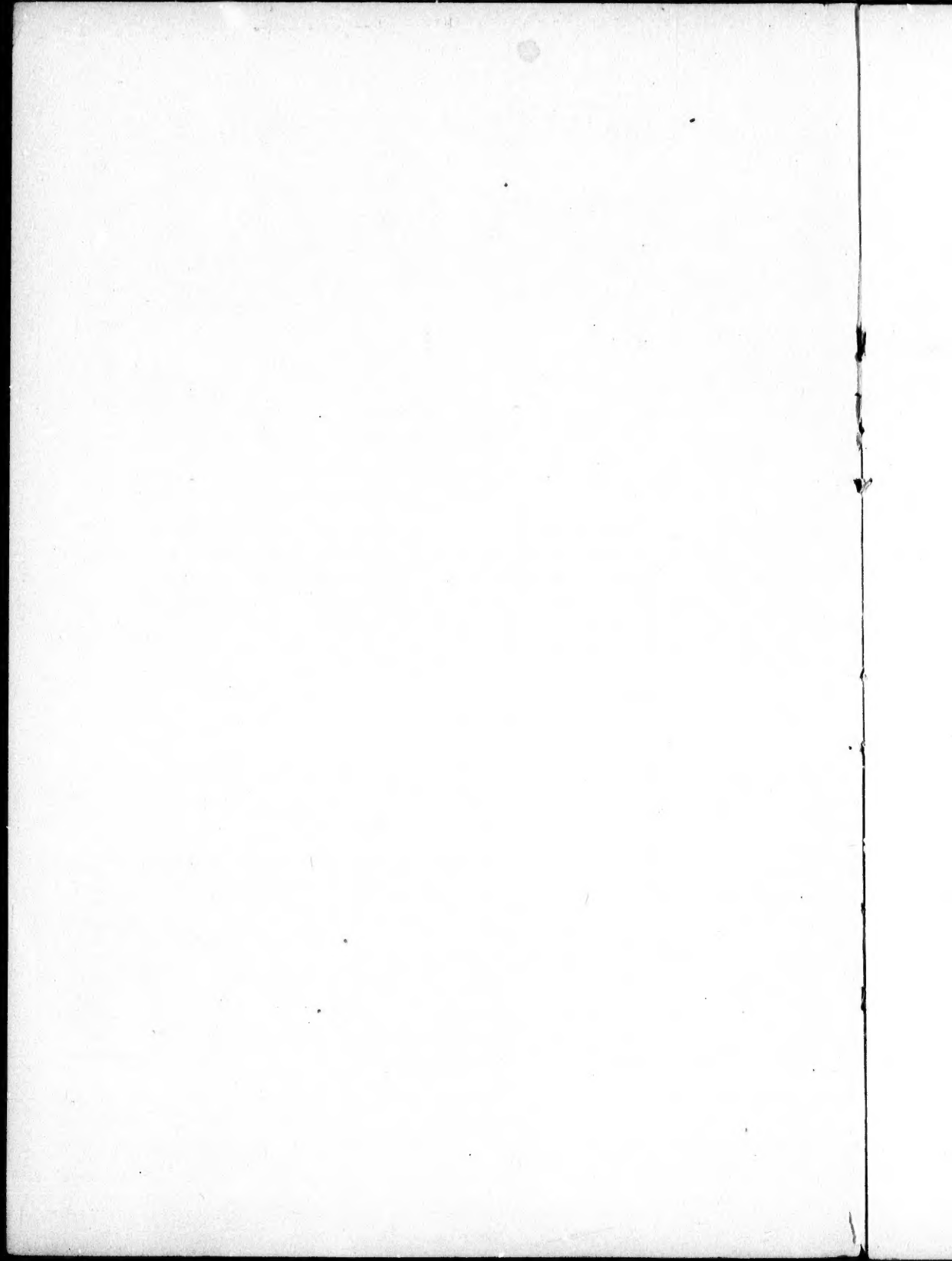
Reprinted from the Montreal Medical Journal, January, 1891.

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SOME CARDIAC PHENOMENA OBSERVED IN TYPHOID FEVER.*

By ROBERT E. McKECHNIE, M.D.,
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Mr. President and Gentlemen:—Through the kindness of Dr. MacDonnell, I am permitted to bring before your notice the two following cases, which were admitted into his wards in the Montreal General Hospital, and so came under my immediate observation as his House Physician. They are two cases of typhoid fever, which would be classed as mild and uninteresting, were it not for certain cardiac phenomena which they presented.

Their histories are as follows:—

CASE I.—Mary M., aged 23, a servant, came into hospital on the 7th day of her illness. With the exception of an attack of smallpox at 2 years of age, she had always enjoyed good health.

On admission, her temperature was 102°; pulse, 100; and respirations, 28. Examination of lungs and heart yielded negative results. The abdomen was tumid with marked tenderness, with gurgling in the right iliac fossa. No typhoid spots were seen and the spleen was not enlarged.

On the 11th day of the fever, typhoid spots appeared. There was no tympanites, and bowels moved only by enemata.

Up to the 18th day the case progressed favorably; the temperature for the previous week rarely going above 102°; the bowels still moving only by enemata; and there being no tympanites or excessive abdominal pain.

* Read before the Medico-Chirurgical Society of Montreal.

On this 18th day, at 6 a.m., the nurse took the temperature as usual, and found it 100°. Half an hour later, noting a great change in the patient, the temperature was again taken and found to be but 98°. The patient was then extremely pale, as though blanched by a hæmorrhage; the pulse about 150 and very weak; the face and extremities cold, and bathed by a profuse cold perspiration. Hæmorrhage being at once thought of, the usual remedies were applied.

By noon the extremities were warmer, and the pulse, although still weak and fast, somewhat stronger.

From this time on to the 28th day (ten days), the patient steadily grew stronger; a slight degree of color returned to the cheeks; appetite was returning, and the temperature had been normal for five days.

The bowels still moved only by enemata, and a close watch was kept on the stools for signs of hæmorrhage; but none appeared, although three large motions had been passed in the interval.

But on this 28th day a fresh syncopal attack occurred. At 7.30 p.m., just after having been made comfortable in bed for the night, which process necessitated a certain amount of disturbance to the patient, she again became suddenly faint, very pale, broke out into a profuse perspiration, and pulse became rapid and feeble. In fact, the pulse was so feeble that the stethoscope was applied over the heart to get the rate. There was then heard over the left ventricle, at the apex, and propagated into the axilla, a loud blowing systolic murmur. Examination failed to find any cardiac enlargement.

Hot applications were applied and stimulants ordered.

Two days afterwards she was much stronger. The murmur, as above noted, still persisted, and in addition a loud systolic murmur was heard at the aortic cartilage, propagated upwards along the vessels of the neck, and also heard across the upper part of the chest, from the aortic cartilage to the left axilla. A slight trace of blood was seen in a stool passed this day, which was the only blood passed throughout the case.

Ten days afterwards the patient had so advanced in convalescence that she was out of bed every afternoon. At this time the murmur could not be heard at the apex at all.

A week later, two days before her discharge, the note in the

case book reads: "The murmur is heard with difficulty at the aortic cartilage, and not at all in the axilla or at the apex."

The second case is entirely different to the preceding, but still presents a similar problem to be solved.

CASE II.—Annie M., aged 21; a general servant, lately out from Ireland, was admitted to hospital on the 6th day of the fever.

When a child she had had measles, scarlet fever, chicken pox, and whooping cough; since when she had always had good health. But the past four months she had found herself breathless on exertion; dizzy at times; and troubled with palpitation.

On admission, she was found to be in a good state of nutrition. Her face, the mucous membranes of the mouth, and the surface of the body generally, were very pale. There was visible pulsation of the carotids; and under the finger nails, and through the mucous membrane of the lips, could be seen a capillary pulse. The abdomen was tumid, with no complaint of pain on palpation; the bowels constipated; the tongue of the ordinary typhoid type. No spots were visible. The superficial area of cardiac dulness was enlarged, measuring $3\frac{1}{2}$ inches transversely, with the apex beating $\frac{1}{2}$ inch inside and under the nipple. Over the upper part of the sternum could be heard a systolic blowing murmur, propagated up the neck, and across the upper part of the chest to the left axilla; but not heard at the apex.

The pulse was semi-collapsing, 102 to the minute, regular in volume and rhythm; temperature, 103° ; respirations, 24. Examination of lungs found them normal. The liver measured 3 inches vertically in the right mammillary line. The spleen was not enlarged.

Three days later, characteristic typhoid spots began to appear; and the next day the spleen was noted as enlarged. But, on the third day after admission (the 9th of the fever), a faint systolic murmur was heard at the heart's apex. It increased in strength for a day or so, becoming quite marked, and co-existing with the original murmur. This persisted for some days. The first sound of the heart was also almost inaudible. The case ran a favorable course and normal temperature was reached on the 20th day.

On the 26th day, the patient convalescing rapidly, I find this note: "Area of cardiac dulness remains as on admission; the apex also where then found. But the systolic murmur then described as being heard over the upper part of the sternum, across into the axilla, up the neck, and the additional one, discovered three days later, at the apex; now at times cannot be heard at all, sometimes can be heard faintly at the aortic cartilage, sometimes at the apex."

Three weeks later, the patient was still in the hospital, having been detained by an abnormal temperature, which appeared after she had been given solid food. At this date, no trace of a murmur could be heard in any of the areas where it had been formerly heard.

While these two cases are different in many important points, they possess this feature in common, that, there was a progressive development of some cardiac affection in each. This was shewn, in the first case, by the presence of a murmur where none existed before and an addition in a few days of another marked murmur. In the second case, a murmur was present when the patient first came under observation, but an additional one was developed during the course of the disease. And in each, as the fever ran its course, these cardiac manifestations became less marked, entirely ceasing in one.

If the two cases cited were not typhoid, the inferences and conclusions to be drawn from them would be erroneous; hence, it will be necessary to briefly exclude those diseases which might be mistaken for it, especially those with cardiac complications.

Murchison says that "the various manifestations of tuberculosis constitute the maladies most difficult to distinguish from enteric fever." Fagge qualifies this by saying that "miliary tuberculosis of the lungs may be mistaken for typhoid with bronchitis." As there was no pulmonary involvement this can be at once excluded. Spots, closely resembling those found in typhoid, are described as being found in acute tuberculosis; but Murchison met with only one such case. It was diagnosed typhoid and died, the autopsy revealing the error. The strongest proof that our cases were not tuberculosis is, that both recovered. The same fact will exclude ulcerative endocarditis, and pernicious anæmia with febrile symptoms.

On the other hand, we have the strongest proofs that our cases were typhoid, in a sequence of typical symptoms, completed by the finding of the characteristic typhoid spots.

Murchison says: "When after febrile symptoms of about a week's duration, lenticular rose spots appear in successive crops, the diagnosis of enteric fever is certain, whatever be the other symptoms. Two or three characteristic spots will be sufficient."

Acknowledging the cases to be typhoid fever, the question arises, what, then, was the nature of the cardiac phenomena? We have in the production of cardiac murmurs to take account of the factors engaged; namely, of the blood, of the endocardium, of the myocardium, and of the pericardium. There were no symptoms at all suggestive of a pericarditis, nor would such a disease account for the signs found; so we may dismiss a lesion of the pericardium at once.

A hæmic origin of the murmurs is not so easily set aside. In our 2nd case we have a girl of 21, probably a hard-worked servant, very anæmic, and with a history of having suffered from breathlessness, dizziness and palpitation for four months previously. No recent cause had occurred to bring on these symptoms, unless it were an anæmic state. But, there is strong evidence that there was long-standing valvular disease in her case. She had had scarlet fever as well as measles when a child, and it is quite possible that an endocarditis had complicated one of these diseases, leaving permanent damage. Compensation had been good from that time till, perhaps four months ago, when she first noticed shortness of breath and palpitation. At this date, also, she left home and went to service, hence hard work may have lessened the perfection of compensation. Or mere anæmia may have given these symptoms irrespective of cardiac disease. At any rate, when the patient came under our observation, she had every evidence of long-standing disease of the aortic valves, in an enlarged heart, with pallor of the surface of the body, pulsation of the vessels in the neck, a capillary pulse, a systolic murmur heard at the aortic cartilage, and propagated up the great vessels of the neck.

If it were not that fresh cardiac symptoms were added to the foregoing, this case would not present the interest it does.

Why an anæmic state of the blood, superadded to the changes which take place in the blood in all fevers, would not especially predispose to the development of cardiac murmurs, without any actual cardiac disease, may well be asked.

According to Bristowe, in his article on Anæmia, "the development of abnormal sounds in the heart and blood vessels, independent of organic lesions, is of common occurrence and highly characteristic, a soft systolic murmur is to be heard frequently over the situation of the aortic and pulmonary valve, and along the course of the ascending arch and innominate artery, occasionally at the apex;" while Fagge, in describing different forms of anæmic murmurs, says: "An anæmic murmur of another kind is systolic in rhythm, and is heard over the heart and main arteries. It is usually loudest at the base, and it often seems to be traceable along the pulmonary artery, rather than along the aorta. . . . Whether an anæmic murmur is ever localized at the apex, I am not sure. . . . The basic systolic murmur often has a rough, harsh quality, suggestive of anything rather than a functional origin."

Fagge, therefore, doubts an anæmic murmur ever being localized at the apex. But in both our cases this occurred. In this 2nd case which we have been discussing, the murmur was developed under observation, and the characters of it suggested regurgitation, rather than that it had a hæmic origin. In the 1st case, the onset of the cardiac manifestations was too sudden to be from a hæmic cause. In both cases, the distribution of the murmurs, together with their intensity of sound, pointed to an incompetency of the valves from some cause. "For an anæmic murmur is usually loudest at the base, and it often seems to be traceable along the pulmonary artery rather than along the aorta." So, while anæmia itself cannot be excluded from the cases, it of itself is not sufficient to account for the collapse in the one case, or the physical signs in either.

That an endocarditis may have been the cause also needs investigation. It is a complication which may be looked for in any of the specific fevers, and slight degrees of it are found in typhoid oftener than commonly supposed. Jenner noted it in three cases out of sixteen, while, according to Hoffman, the endocardium is often opaque and thickened, owing to a fatty degeneration of its lining epithelium; and in several instances

he found recent endocarditis, with vegetations on the aortic and mitral valves.

Bristowe says a subject of endocarditis remains liable to fresh attacks of the inflammation, and there is every probability to suppose that Case 2 was a former subject of endocarditis. I think there is some proof, that not only were the aortic valves then affected, but also the mitral, for a contracted liver of three inches vertical dulness was noted, which would be accounted for by chronic mitral incompetency. If this be the correct view, then compensation had later on improved, so as to be about perfect, but was again lost during the course of the fever to an appreciable extent, and regained as convalescence advanced. The cause of this slight loss of compensation and recovery will be discussed later on, and is not an endocarditis.

But endocarditis cannot be thus easily dismissed, for certain points are strongly in its favor. Thus Bristowe says: "If in the progress of any one of those diseases of which endocarditis is a common complication, we detect a cardiac murmur which had not previously existed; or if further observation proves this to be a permanent phenomenon; or if changes in it indicative of increasing mischief take place; or if additional murmurs become developed, we cannot reasonably doubt that endocarditis is present." And as further support he says: "We must not forget that direct murmurs due to granulations occasionally disappear."

Now, in regard to these statements, we must note that Bristowe speaks of those diseases in which endocarditis is a common complication, which statement lessens the value of the observation when we try to apply it to typhoid; for endocarditis is not a common complication of it. Bristowe, in making this statement, was carefully weighing evidences and probabilities, which were of value only in the class of cases he was thinking of, so that having taken typhoid out of that class, the probabilities of endocarditis being present are reduced, unless new evidence is forthcoming.

The fact that our murmurs lessened and disappeared is, of course, not at all contrary to the view that endocarditis was their cause; but the fact that they lessened and disappeared coincidently with the return of strength, as convalescence advanced, strongly suggests another cause, which either will

account for the phenomena under discussion, or will act as a factor, in conjunction with the causes already discussed, to account for them. This is disease of the myocardium.

Degeneration of the muscle cells of the heart walls is common to many different fevers. According to Murchison, "Louis found marked softening, sometimes associated with thinning of the walls, in 15 out of 47 cases of typhoid. Jenner found the heart soft and flabby in 6 out of 11 cases; . . . and Hoffman has found either waxy or granular degeneration of the muscular tissue of the heart in 103 out of 159 cases."

Hutchinson, in Pepper's System, observes that "the heart, in common with the other muscles of the body, suffers from both forms of degeneration, but the granular form appears to be more common than the waxy. In protracted cases it is usually much softened. . . . Upon minute examination, the degeneration is found to have taken place in patches, the diseased fibres being found alongside of others which have scarcely undergone any alteration. These patches are especially common in the papillary muscles of the mitral valve, a fact which explains the occasional presence of systolic murmurs in typhoid fever."

In another place he also says: "Degeneration of the muscular tissue of the heart is probably present in some degree in every case of typhoid fever, being, of course, most marked in the severest cases."

From the foregoing, it is evident that a weakening of the cardiac wall must be looked for in every case of typhoid, and is a factor too important to be overlooked in hunting for an explanation of the signs found in our cases. That we have evidence that this parenchymatous degeneration may occur early in the disease, is seen in the tremors of the tongue, an early observable fact. These tremors, according to Fagge, are due to this cause.

Now, anæmia itself is a common cause of parenchymatous degeneration of the muscle wall of the heart, and may lead to so much weakening, with dilatation, as to give cardiac murmurs which are not hæmic. Both our cases were anæmic on admission, hard-worked servants. So we have a two-fold reason for supposing the heart-walls weakened, fever and anæmia.

According to Osler, "impaired nutrition of the heart-walls, from degeneration or inflammation, may lead to such a diminution of the resisting power that dilatation readily occurs. In fevers, the loss of tone due to parenchymatous degeneration or myocarditis, may lead to a condition of acute dilatation which may prove fatal. It is a well recognized cause of death in scarlatinal dropsy, and may occur in rheumatic fever, typhus, typhoid, erysipelas, etc. . . . In anæmia, leukæmia and chlorosis, the dilatation of the chambers may be considerable. . . . Under any of these circumstances, the walls may yield with normal blood pressure, or if increased tension is present, the effect is the more readily produced."

Again, he says: "Dilatation with thinning is often the result of an acute process met with in the fevers. . . . The auriculo-ventricular rings are often dilated, and there may be an inch and a half, or even two inches, of increase in circumference. . . . Great dilatation is always accompanied with relative incompetence of the valves, so that free regurgitation into the auricles is permitted."

And again: "In anæmia and fevers the temporary dilatation may undoubtedly pass away with the improvement of health."

In connection with weakening of the cardiac walls, an important sign is a feeble or imperceptible first sound. This is found in all forms of weak heart, the fatty, as well as that the subject of febrile degeneration. In our 2nd case this sign was marked; but I find no note in the case book on this point in reference to Case 1.

The presence of an apex murmur, also, may be a proof that there were weakened cardiac walls with some dilatation. To quote from Loomis' article in Pepper's System: "Mitral regurgitation may occur independent of valvular disease, from displacement of one or more of the segments of the valve, the result of changes in the papillary muscles, chordæ tendinæ or the ventricular walls. It may also occur in extreme anæmia, or from relaxations of the papillary muscles, etc." In another place he says: "It may occur in conditions of extreme anæmia, or where there is degeneration of the walls of the left ventricle."

Returning to our two cases, there is no need to try to adapt our explanation to fit these two, merely because one ward con-

tained them at the same time. I do not think a single explanation would satisfy the symptoms noted. So, with the foregoing extracts, culled from various sources, we are in a position to attempt to clear up the subject. In Case 2, my opinion is, that a previously anæmic condition coupled with the degenerating influences of the fever, led to an early weakening of the heart wall. The fact that as convalescence advanced, both aortic and mitral murmurs disappeared, leads one to judge that before her illness, when she was strong, there were no murmurs; and that, even before admission, a gradual dilatation of the left cavity of the heart had begun, shewing itself in disclosing a long hidden aortic lesion. In a few days more the cusps of the mitral valve allowed regurgitation from a continuation of the same process. This idea is merely the reverse of what is seen in most cases of endocarditis, as in rheumatism; for there is weakening of the myocardium in it, as in all fevers. In it, as convalescence progresses, the murmurs are reduced in intensity, and often disappear entirely, long before the soft granulations of the valves could have been altered by cicatricial changes. If, then, the restoration of tone to the ventricular wall does in many cases lessen or cause to disappear murmurs due to valvular lesions, then, in the present case, a loss of tone in the ventricular wall of a heart with valvular lesions can cause to re-appear murmurs which formerly existed. And as proof that this is the rational explanation, we find, as convalescence advanced, as tone returned, the murmurs vanished.

Such is my explanation of the cardiac phenomena observed in Case 2; but we will have to look elsewhere for the causes at work in Case 1.

In an hypertrophied heart, as in Case 2, slight dilatation could occur sufficient, perhaps, to allow of the re-appearance of former murmurs; and yet, the dilatation be not readily made out by the Clinician, unless specially watching this one point. But in Case 1, with a heart of normal size on admission, with no evidence of organic disease, dilatation sufficient to cause regurgitation would so enlarge the cardiac area of superficial dulness as to be easily recognizable. But the notes of the case say distinctly that there was no enlargement of the heart at any time during the course of the illness.

Nor could dilatation account for the systolic aortic murmur. An enlargement of the aortic orifice would lessen instead of increase the resistance to the passage onward of the blood. The cause of this murmur must then be either hæmic or endocardial.

Loomis, in differentiating murmurs due to lesion of the aortic valves, from hæmic murmurs heard in somewhat the same areas, says: "Anæmia produces a murmur that is heard loudest in the carotids and is accompanied by a venous hum. In aortic disease, the murmur has its maximum intensity at the second sterno-costal articulation of the right side and is not accompanied by a venous hum." In this case the point of maximum intensity was at the aortic cartilage, and there was no venous hum, although it had been especially looked for.

I found several references to systolic apex murmurs being met with in fevers; but Loomis, while speaking of these, says: "There are no symptoms of obstruction present with febrile murmurs, while they are frequently present in endocarditis." Thus, he would at once exclude an aortic systolic, which is an obstructive murmur, from the febrile class. Nothing remains, then, but to acknowledge endocarditis as the cause, and if so, then the mitral murmur first heard must also be from the same. We have, then, a case of endocarditis in typhoid, following the usual course, attacking first the mitral valves, and a few days later the aortic.

What reference this endocarditis had to the symptoms of collapse, described as having been seen on the 18th day of the fever, and a second time on the 28th day, needs inquiring into.

We can imagine a myocardial cause at work, which would so weaken the cardiac walls as to cause a muscular incompetency. But such a state would declare itself by dilatation of the organ, which we know did not occur; and also by embarrassment of the pulmonary circulation. But this, too, was looked for and not found. If the heart were the organ mainly at fault, we would have found proof by physical signs. The valvular lesion could not have been the cause any more than a muscular incompetency, for it also would have shewn itself by engorgement of the pulmonary circulation.

Where, then, was the greater part of the blood? It was not

in the cutaneous structures or the extremities, as evinced by the extreme pallor and coldness of surface. It was not in the bowel, where it had been diagnosed to be, for the stools proved the negative. It was not in the chest, for the heart had too little blood for the needs of the circulation, and the lungs were not engorged. It must, then, have been stagnating in the dilated veins of the splanchnic area, which area is capacious enough to hold all the blood in the body.

Bristowe, in discussing collapse, accounts for the depression of temperature, as far as regards the face, limbs and other exposed parts, by the comparative failure of circulation in them. "But," he says, "that this is not the sole cause of that depression is obvious from the fact that the internal temperature, instead of rising, as under such circumstances it should normally do, itself tends to diminish, and sometimes diminishes rapidly. It is clear, indeed, that there is throughout the organism a more or less complete arrest of those disintegrating processes upon which the maintenance of the temperature of the body depends, and presumably also, a more or less complete arrest of those vital processes, with which these latter are intimately interwoven."

That he considers this due to nervous action is plain, from his concluding paragraph on this subject.

"We have pointed out the not infrequent dependence of collapse or syncope on affections of the mind, and on many other conditions, which can be operative only through the medium of the nervous system; and we have enumerated the various phenomena referrible to the nervous system, which attend and characterise a large proportion of cases. These facts are sufficiently suggestive. But when we look a little more closely into the matter, and consider how many different causes of different operation, equally produce the same collective phenomena of collapse; how rapidly these phenomena come on, and how universally the organism is affected by them; how impossible it seems that a smash of the leg, a perforation of the bowel, or an agony of terror, should directly arrest the chemical changes going on throughout the organism, and so reduce the temperature of the body, or should directly influence the action of the heart and arteries; it is impossible to doubt (what many other considerations tend to prove), that all the phe-

nomena of collapse are directly traceable to the operation of the nervous system, not, however, of the brain or cord, but of that department, namely, the sympathetic, which presides over circulation, nutrition and the functions of the various organs, including those of the brain itself."

In the case under observation, we have no crushed leg or violent emotion to give the initiative impulse to the sympathetic system; but we have in typhoid a lesion, of that area most richly supplied by the sympathetic, an ulcerative process in the intestine, a reflex of whose irritation is the factor looked for.

In the *Lancet* of 1880 I found a short article on "Sudden Death in Typhoid Fever." The author says: "The occurrence of sudden death in typhoid fever, without any hæmorrhage or perforation to explain it, has been variously accounted for. According to Dieulafoy, it is always due to syncope, a reflex result of the intestinal lesion."

Thus I account for the collapse observed in Case 1. It was due to a reflex of the intestinal lesion, acting through the sympathetic nerves and manifesting itself by a dilatation of the veins in the splanchnic area. The withdrawal of a large quantity of blood from the general circulation, or the stagnating of it in this manner would produce the same constitutional effect as an actual hæmorrhage, namely, collapse.